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ORIGINAL PAPERS

BREATHING EXERCISES: ARE THEY OF VALUE?*

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DURING the past few years a series of publications by competent respiratory physiologists have decried breathing exercises as being of value in emphysema. If this is true, I agree that one should abolish the waste of time and energy and the expense of physiotherapists engaged in this work. In spite of the negative findings of the scientific observers, however, I think we should be sure of our ground from the clinical aspects before we decide to discontinue these measures.

I should like to restate my conceptions based on 25 years of practice with asthmatics and bronchitics: these concepts may be elementary and even incorrect, but the air must be cleared on fundamentals.

MECHANICS OF BREATHING

In natural breathing the inspiratory mechanism is stronger than the expiratory. The diaphragm is a powerful inspiratory muscle, and as a result of bronchospasm or repeated coughing it may easily develop increased tone which may persist indefinitely. To a lesser extent the same applies to the intercostals, scalenes, and other accessory muscles.

Reflex contraction of the bronchial muscles occurs at the beginning of expiration, so that a check-valve mechanism resulting in over-inflation of the lungs is easily set up by factors which narrow the lumen of the bronchioles —for example, contraction or failure of relaxation of the muscle, swelling of the mucosa, or secretion from the glands.

* A symposium presented at a meeting of the Section of Physical Medicine of the Royal Society of Medicine, November, 1957.

Normally expiration takes place by the elastic recoil of the lungs. This is interfered with in the presence of bronchospasm or emphysema: active contraction of the abdomino-thoracic muscles is necessary to prevent over-inflation under these conditions. The habit of breathing with over-inflation of the lungs is easily acquired, with persistent increase in the residual air and with diminished mobility of the chest wall and diaphragm. In the absence of infection this over-inflation does not cause emphysema, but it may be associated with dyspnoea. When organic emphysema has occurred this over-inflation may be a factor in the dyspnoea.

During quiet respiration a large number of alveoli are either closed or not functioning to maximum capacity; the dilution of tidal air in the functional residual air must be expected to affect the rate of gas-mixing. Buxton (1957) has shown in a group of normal students that after a deep breath the functional reserve air is increased from a mean of 3·65 to 4·28 litres, and the ratio of residual capacity to total lung volume is increased from a mean of 28·6% to 37·2%. In a series of patients with emphysema the mean functional residual air was high at 4·75 litres, and after deep breathing there was no material increase in the functional residual air, indicating diminished respiratory reserve.

Dyspnoea is a symptom, but anxiety and nervous tension increase the tone of the overacting inspiratory muscles and increase bronchospasm. The latter is present in the majority of cases of "chronic bronchitis", as shown by the improvement in respiratory function tests after inhalation of bronchodilators. Physical and mental relaxation should be encouraged as a therapeutic measure.

When a patient with bronchospasm is encouraged to expire fully, increased wheezing and coughing is to be expected. I believe that when these expiratory efforts are gently continued there may be a reflex action on the bronchial glands resulting in secretion of mucus, so that the patient is able to expectorate small plugs, with relief of wheezing.

On fluoroscopy during an attack of asthma the diaphragm is usually seen to be low and moving poorly; when the patient is asked to expire forcibly there is little rise in the level of the diaphragm. When the attack has ceased the diaphragm is seen to be higher in the chest, the movements are increased, and on forced expiration the level of the diaphragm rises markedly. A low, flat, contracted diaphragm with a poor range of movement is present in greater or less degree in the majority of patients with simple over-inflation or with emphysema.

Wade (1954) describes a method for accurately recording the movements of the chest wall and diaphragm. He concludes that even a trained physiotherapist has little voluntary control over the movements of the diaphragm, and

that movements of the anterior abdominal wall do not indicate the extent of diaphragmatic movements. While agreeing that the methods used are excellent and that the investigations were carefully carried out, I cannot quite accept his conclusions. From my own observation on many hundreds of cases I am quite sure that a person who has been successfully trained by a competent physiotherapist can move the lower chest wall to a greater degree than someone not so trained, and, further, that by this movement of the lower chest and epigastrium the range of diaphragmatic movement, as seen on fluoroscopy, is greater also. I had thought that these trained patients, when asked to breathe with the upper thorax, moved the diaphragm less than when they were told to breathe "abdominally". Wade's explanation that the vertical movement of the chest wall in full inspiration gives a false impression of the range of diaphragmatic movement may be true.

In my practice, anyway, I have found that a patient who has acquired breathing control usually has a good lower chest expansion, and on fluoroscopy during quiet respiration the diaphragm is moving smoothly, well above the position of full inspiration. Of my asthmatics who have acquired good "breathing control" only 10–15% have failed to improve as regards the asthma.

ROLE OF PHYSIOTHERAPY

What are the possible benefits to be derived from physiotherapy? In addition to breathing exercises the physiotherapist teaches the patient to maintain a good posture, to discourage useless cough, and to relax both mentally and physically. The thoracic deformity caused by wrong breathing should be prevented or decreased if treatment is started at an early stage.

The principle of "athletic training" should be used by graded athletic exercise to the limit of tolerance in the young asthmatic, and within reason for older patients. Many are afraid to take exercise because they notice increased wheezing and dyspnoea, but yet they are much improved by graded athletic exercise.

The modern physiotherapist is a well-trained person with experience in dealing with patients, and with time to give them individual treatment, to listen to them, and to encourage them. The competitive element in class work acts as an incentive. There is little doubt in my mind that a physiotherapist needs some experience in chest work, and that one with personality and enthusiasm gets better results than one without these attributes.

I shall not discuss the inspiratory type of breathing exercises used after thoracic surgical procedures, such as resection, thoracoplasty, etc., nor the routine breathing exercises given before abdominal operations.

ASTHMA

I define asthma as a clinical syndrome marked by periodic dyspnoea with expiratory wheezing and caused by temporary narrowing of the bronchioles throughout the lungs. In my opinion, the two most important causes are allergy and infection, alone or in combination. Nervous tension caused by the asthma is an important factor in determining the severity of the attacks.

There are so many variables in the asthmatic—local allergens, seasonal influences, infections, nasal diseases, nervous factors, and so forth—that the results of any one treatment must be assessed with a follow-up of at least two and preferably five years. It is obviously difficult to get a group of patients to attend regularly for any one treatment and for observation over a number of years, and therefore such a long-term follow-up must consist of selected rather than consecutive cases.

TABLE I

RESULTS OBTAINED IN 20 ASTHMATIC MALES (INFECTIVE GROUP)

Results	At 1 year	At 2 years	At 3 years	At 4 years	At 5 years	At Last Follow-up 6-22 years	Other Treatment
Cured*	0	2	4	4	5	4	–
Excellent	5	4	1	1	0	0	–
Very good	2	1	0	2	0	1	–
Much improved ..	4	5	3	1	0	0	1
Failed	9	8	10	6	8	5	9
Died	–	–	1	3	3	(4)	–
Lost	0	0	1	3	4	10	10
Total	20	20	20	20	20	20 (4 died)	20

*Cured=symptomless for at least twelve months and not having any kind of treatment.

A series of asthmatic patients was investigated at King's College Hospital from September, 1932, to December, 1935, by myself with Dr. Marjorie Gillespie and Dr. E. I. Jones, and treated by breathing exercises as the main form of therapy (Livingstone, 1957). We excluded those who failed to attend, those who changed their environment or occupation, those who removed allergens from the bedroom, and those who had other therapy such as nasal operations, inhalers, vaccines, etc. The number that were excluded for various reasons is considerable, but I believe that the residual 110 cases fulfilled the requirements and were carefully and conscientiously assessed, and followed up for periods ranging from two to twenty-three years.

Table I gives a summary of the results obtained in a group of 20 male asthmatics in which infection with recurrent or persistently purulent sputum seemed to play a major part, whether there was a background of allergy or not. I have selected this group as being the worst type of asthma to treat. At five years only 5 remained "cured", 8 failed, and 3 had died. The majority of the failures did not respond to other forms of therapy. In Table II are given the results in a similar group of allergic males, by chance the same number as the

TABLE II

RESULTS OBTAINED IN 20 ASTHMATIC MALES (ALLERGIC GROUP)

Results	At 1 year	At 2 years	At 3 years	At 4 years	At 5 years	At Last Follow-up: Breathing Exercises Only	At Last Follow-up: Other Treatment
Cured	1	4	10	10	10	2 (1 died)	2
Excellent	4	6	1	–	–	–	–
Very good	6	2	1	1	–	3	–
Much improved ..	3	2	2	–	–	–	1
Failed	6	4	3	4	4	6 (1 died)	6
Lost	–	2 (2 failed)	3 (3 failed)	5 (4 failed)	6 (4 failed)	9 (3 failed)	–
Total	20	20	20	20	20	20	

TABLE III

RESULTS OBTAINED IN INFECTIVE (BRONCHITIC) GROUP

Results	At 1 Year	At 2 Years	At 3 Years	At 4 Years	At 5 Years	At Last Follow-up 6-22 Years	Other Treatment
Cured	2	7	13	12	9	8 (1 died)	2
Excellent	9	7	5	1	–	–	–
Very good	3	5	–	1	2	1	1
Much improved ..	9	6	5	4	–	1	4
Failed	25 (1 died)	21 (1 died)	21 (1 died)	20 (3 died)	19 (4 died)	15 (6 died)	16 (6 died)
Lost	–	2 (1 failed)	4 (2 failed)	10 (4 failed)	18 (7 failed)	23 (12 failed)	–
Total	48	48	48	48	48	48	

infective group. At five years 10 (50%) are "cured", showing the better prognosis without infection.

The results are shown in Table III in a total of 48 men, women, and children with infective asthma. At five years only 9 are "cured", 26 failed, and 4 have died (8%). Table IV gives the results in 62 men, women, and children with allergic asthma. At five years 19 (about one-third) are cured and 38 (about two-thirds) have failed. In the follow-up 13 had failed to respond to other treatments and 13 had improved.

TABLE IV

RESULTS OBTAINED IN ALLERGIC GROUP

Results	At 1 Year	At 2 Years	At 3 Years	At 4 Years	At 5 Years	At Last Follow-up 6-22 Years	Other Treatment
Cured	1	10	18	20	19	8	2
Excellent ..	12	13	2	–	1	–	–
Very good ..	12	8	6	4	2	4	5
Much improved	17	10	8	2	2	1	6
Failed	20	18 (1 died)	19 (1 died)	13 (1 died)	9 (1 died)	19 (5 died)	13
Lost	–	3 (3 failed)	9 (7 failed)	23 (15 failed)	29 (19 failed)	30 (15 failed)	–
Total ..	62	62	62	62	62	62	

EMPHYSEMA

I had thought that in assessing the results of treatment emphysema would be a much simpler condition to deal with than asthma; in this I was optimistic. The diagnosis of emphysema is not a simple matter: is it merely over-inflation or is it organic emphysema, or a combination of the two, plus bronchospasm with a dash of smoker's catarrh and a variable degree of infection? If organic emphysema, is it mild or advanced? The answer depends largely on the physician's mood at the moment. Research on the subject is complicated, as are the respiratory function tests which are said to make the matter clear. Given an extra 20 cigarettes, a mild bronchial cold on a sunny day in June or a foggy afternoon in November, and even the respiratory physiologist is left scratching his head.

On *a priori* grounds I had thought that if it were possible to teach a patient, diagnosed as having "emphysema", to acquire a more mobile diaphragm and

chest wall and to decrease over-inflation the respiratory function might be improved, depending on the respiratory reserve which remained. I have accepted the clinical impression that if a patient, breathless on one flight of stairs, completed his treatment by physiotherapy by climbing 70 stairs to the department on the roof of the Brompton Hospital he had acquired some benefit. Many patients do in fact do this, but I freely admit that I don't know how many or whether in fact their story of exertional dyspnoea before treatment is true.

K. M. Hume (personal communication) has recently investigated at the Brompton Hospital a number of patients labelled "emphysema" and treated for three months by the physiotherapist at that hospital. Of 30 patients selected only 11 fulfilled the conditions laid down; in none of these was there any marked improvement in the series of function tests performed by Dr. Hume. When asked their views two patients said they were "much better", five said they were "definitely better", and two were unchanged.

In spite of this, as a clinician I should be very sorry if physiotherapy was given up in these cases. I am quite convinced that a proportion of patients who acquire "breathing control" do in fact improve their respiratory function. I agree that further critical observations extending over two to three years are required on a large series of cases treated by physiotherapy and compared with a similar number of control cases treated with a placebo.

ACKNOWLEDGMENT

I am indebted to the Editor of the *Transactions of the Medical Society of London* for allowing me to use Tables III and IV.

REFERENCES

BUXTON, R. ST. J. (1957) *Brit. J. Tuberc.*, **51**, 1.
LIVINGSTONE, J. L. (1957) *Trans. med. Soc. Lond.*, **72**, 108.
WADE, O. L. (1954) *J. Physiol.*, **124**, 193.

II—D. A. BREWERTON

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THOSE working in the field of physical medicine are interested in the restoration of function by exercise therapy, but most would agree that the major responsibility for chest disorders must fall to physicians, surgeons, and general practitioners. Tests of respiratory function have become so complicated that it is difficult for anyone in our specialty to do research on this subject, but this does not mean that we have no contribution to make. In the first place, the techniques of

breathing exercises were mainly designed by specialists in physical medicine co-operating with chest physicians and surgeons; and to-day many of us help to teach physiotherapists about these techniques and their purpose, and help them to maintain a high standard of treatment. In the light of current medical research we are expected to discuss any changes in medical opinion with our senior physiotherapists. Finally, we have an important administrative task in seeing that an adequate number of physiotherapists are available to carry out any treatment which can be shown to be of value.

Looking at the problem from this viewpoint, few of us are satisfied with the present position. We know that breathing exercises came into vogue twenty years ago without proof of their value. Now more and more physicians and research workers are becoming critical, so that Donald (1953) can state: "Most of us are inclined to believe that breathing exercises are a convenient placebo, and an indirect form of psychotherapy in respiratory conditions where little else can be offered." Despite this growing attitude, these exercises are being prescribed more commonly than ever before—though often with criticism instead of guidance for the physiotherapist, and with indifference instead of enthusiasm for the patient.

WADE'S CONCEPT OF RESPIRATION

Wade (1954) has cleared away many misconceptions with his studies of the thoracic cage and diaphragm during normal respiration. Normal inspiration combines two movements of the thoracic cage—expansion of the ribs, which continues throughout inspiration; and "lifting of the sternum", which normally occurs only towards the end of deep inspiration and is associated with slight extension of the dorsal spine. With voluntary effort these two processes can be separated, so that it is possible to inspire deeply by expanding the chest without lifting the sternum, or to lift the sternum without inspiring at all. Under voluntary control the ribs can be moved more on one side than the other by slight lateral flexion of the dorsal spine; and the abdominal wall can be moved in and out without influencing movements of the diaphragm. Armed with these simple manœuvres, it is possible to give the appearance of doing "costal breathing", "diaphragmatic breathing", "lower costal expansion", or "lower costal expansion confined to one side". "Costal breathing" involves lifting the sternum during inspiration. To change this to so-called "diaphragmatic breathing" it is only necessary to inhibit the lifting of the sternum and push forward the abdominal wall during inspiration. "Lower costal expansion" is like any other deep inspiration, except that lifting of the sternum is inhibited; and it can be confined to one side by adding slight lateral flexion of the dorsal

spine. But, according to Wade, it is not possible to influence either the amount the ribs expand, or the excursion of the diaphragm, except by controlling the amount of air inspired. Apparently these three factors go together; the volume of air inspired, the rib expansion, and the diaphragmatic movement are proportional throughout inspiration. This relationship is not influenced by movements of the dorsal spine or of the abdominal wall, and it cannot be altered by training.

Wade has also explained some apparent anomalies found when observing diaphragmatic movement on a fluorescent screen. Sometimes during inspiration the diaphragm is seen to rise instead of fall, and this is described as a form of "paradoxical movement of the diaphragm". It is due to extension of the dorsal spine and lifting of the sternum which raise the diaphragm in relation to the X-ray tube. To appreciate the true descent of the diaphragm during inspiration it is essential to measure diaphragmatic movement in relation to the chest wall. This is important, for a patient who is accustomed to lifting his sternum and is then taught to inhibit this movement may give the impression that his diaphragmatic excursion has improved. Sinclair (1955) has shown that this is an artefact and that, provided diaphragmatic movement is measured in relation to the chest wall, the excursion of the diaphragm is not altered by breathing exercises.

BREATHING EXERCISES IN EMPHYSEMA

The effect of breathing exercises on pulmonary emphysema has been investigated by several workers, who have failed to show convincing improvement in vital capacity, diaphragmatic excursion, maximum breathing capacity, or expiratory flow rate. But these are mainly measurements of structural alteration, and alone they do not prove that breathing exercises are without value.

Miller (1954) and Campbell and Friend (1955) have investigated the effects of emphasizing the expiratory phase of respiration. This means breathing more slowly, more deeply, and below the individual's normal resting respiratory level, so that the residual air is reduced. When breathing in this way the minute volume is not increased, as the respiratory rate is so slow, but there is more effective alveolar ventilation due to the reduction in residual air. Miller found that this method of breathing substantially increased the arterial oxygen and reduced the plasma CO_2. This would be excellent were it not for two things. First, this type of breathing involves a conscious effort, and it is difficult to teach a patient to do it for prolonged periods, even at rest; secondly, as Campbell and Friend point out, patients tend to abandon this way of breathing during physical activity. Nevertheless it is not impossible to maintain it under stress, and Miller was able to demonstrate an appreciable improvement in arterial oxygen and plasma CO_2 by active expiration during physical activity. Miller

(1954) and Campbell and Friend (1955) agree that patients made breathless by exertion were relieved of their dyspnoea more rapidly by breathing the way they had been taught.

So it seems there is something to be said for teaching patients with pulmonary emphysema to concentrate on active expiration, thereby reducing their residual air. At least this gives them a method of ridding themselves of excess CO_2 and of overcoming dyspnoea after exercise. But if this is to be taught efficiently all unnecessary frills must be left out. I agree with Campbell and Friend, who say that "pushing the abdomen in and out adds greatly to the patients' difficulty in learning the exercises". Extraordinary answers are obtained by questioning patients about breathing exercises they have learned a year or two previously—"That's where you breathe in through your nose and whistle out through your teeth", or "You lie on your back and lift both legs in the air". It is important that this type of respiration be mastered, not only when sitting or walking, but also during strenuous physical exercise. As Dr. Livingstone has said, we first teach our patients to control their respiration at rest, and then progress them through increasing activities until they attempt to control it under considerable stress.

BREATHING EXERCISES IN ASTHMA

Bronchial asthma, unfortunately, has not been studied in the same way as emphysema. During and after asthmatic attacks prolonged active expiration is advised in the belief that it reduces the air that has been trapped by bronchial obstruction, and most of us recommend this form of exercise. But Wade's work must make us question attempts to teach control of diaphragmatic and lower costal movements. If we accept his work, then we must abandon these two aspects of treatment, not only because they are without value, but because they confuse the patient. If prolonged active expiration is the important factor, then it alone should be taught.

Some patients can abort attacks of asthma by adopting the method of breathing they have been taught—of this there can be no doubt. We are all familiar with the asthmatic at the onset of an attack, panting with frenzied, inefficient little breaths, pausing only for an occasional puff at his hand inhaler. There is a lot to be said for teaching this type of patient something that he can do for himself. As the attack starts he should try to breathe slowly, deeply, and with an emphasis on expiration. He should also expire against pursed lips to reduce the amount of wheezing. The effect of this regimen may be largely psychological, but it is no less valuable for that.

We have to turn back over twenty years for evidence of results obtained from the use of breathing exercises in asthma. Livingstone and Gillespie (1935)

treated 75 patients with breathing exercises and 50 patients with the routine measures then employed—the avoidance of dust, feathers, and other allergens, the use of vaccines for bronchial infection, and hydrochloric acid for those with achlorhydria. They found that an equal percentage of patients in both groups said that they were better and that the number of their attacks had diminished. Unfortunately, at that time a formal, controlled trial was not possible, and no comparable study has been made since. McNeill and McKenzie (1955) have failed to demonstrate any improvement in respiratory function in over 50 asthmatics and bronchitics treated with breathing exercises.

BREATHING EXERCISES IN SURGERY

Pre- and post-operative breathing exercises are also in need of reconsideration. Most doctors agree that in the prevention and correction of post-operative pulmonary collapse the important physical measures are postural drainage, efficient coughing, and deep breathing. We appreciate the excellent way in which physiotherapists can help with these measures, but then we find that they are also requested to teach their patients "breathing exercises"—every day up and down the country hundreds of patients awaiting abdominal operations are taught to control their diaphragms and to expand their bases by lower costal expansion. We may well ask whose task it is to decide on the value of this treatment. Does its continuation simply depend on the division of responsibility between the surgeon, the anaesthetist, and the physical medicine consultant?

Palmer and Sellick (1953) describe a most useful study. They compared the incidence of pulmonary collapse in two groups of patients who had undergone operations for the repair of inguinal hernia or for partial gastrectomy. Of 90 patients treated with postural drainage and isoprenaline inhalations there was segmental atelectasis in 8·9%; while in 90 patients treated with breathing exercises the incidence was 43·3%. Study of a further 160 patients showed that, for good results, isoprenaline inhalations must be combined with postural drainage and percussion. Inhalations alone or postural drainage alone were not effective.

Physiotherapy before and after chest surgery is now widely used. It is impossible to assess its value accurately at present, but it is my personal impression that experienced physiotherapists have an essential contribution to make in this field.

CONCLUSION

In conclusion, having reviewed this subject and discussed it with many people, I am left with three impressions: (1) that we have a lot to learn before we can evaluate breathing exercises satisfactorily; (2) that the information we need

should not be difficult to obtain with research; and (3) that physiotherapists are being asked to do a great deal of work under the twin shadows of partial ignorance and dissatisfaction on the part of the doctors.

REFERENCES

CAMPBELL, E. J. M., and FRIEND, J. (1955) *Lancet*, **1**, 325.
DONALD, K. W. (1953) *Brit. med. J.*, **1**, 415.
LIVINGSTONE, J. L., and GILLESPIE, M. (1935) *Lancet*, **2**, 705.
MCNEILL, R. S., and MCKENZIE, J. M. (1955) *Thorax*, **10**, 250.
MILLER, W. F. (1954) *Amer. J. Med.*, **17**, 471.
PALMER, K. N. V., and SELLICK, B. A. (1953) *Lancet*, **1**, 164.
SINCLAIR, J. D. (1955) *Thorax*, **10**, 246.
WADE, O. L. (1954) *J. Physiol.*, **124**, 1954.

III—A. C. DORNHORST

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[At the meeting at which the preceding papers were delivered I opened the discussion with some remarks from the point of view of a physician interested in pulmonary physiology. In the event these remarks turned out to be mostly irrelevant, and the Editor has kindly allowed me to recast them in the light of the other contributions.]

I SHALL follow Dr. Livingstone in concentrating on the use of breathing exercises in asthma and emphysema. Although chronic asthmatics are often suspected of having emphysema as well, this is not in fact often the case, and so it is practicable as well as desirable to consider the conditions separately.

In emphysema we are dealing with a persisting pathological state, which can now be objectively assessed with fair accuracy; and the dyspnoea, although aggravated by attacks of bronchial infection, usually exhibits a steady basal severity. The task of assessing therapy is therefore reasonably straightforward. Asthma, on the other hand, is characterized by gross and unpredictable fluctuation of disability. There need not be any irreversible changes, and, while objective tests can satisfactorily quantitate the disability at any given moment, they scarcely help the assessment of average disability over a period. It is thus intrinsically more difficult than with emphysema to evaluate therapy.

EMPHYSEMA

Let us consider the simpler problems of emphysema first. Since we are primarily interested in a subjective state—namely, dyspnoea—the simplest course is to ask patients whether they consider they have benefited by treatment. Consistently negative replies would be sufficient to discredit the treatment, but what about positive replies? It would surely be naive to interpret these as

strong evidence of a specific effect: the patients may be responding with gratitude and hope to the interest and enthusiasm of their attendants. It is sometimes asked why one should trouble to analyse the mechanisms that lead a patient to claim improvement—if he says he is better, it is suggested, that is enough. The answer is that if its effect is non-specific the treatment is likely to be extravagant of skilled effort. Moreover, the continued use of ill-founded therapeutic rituals tends to entrench conceptual errors, and hence to hinder progress.

Now, the specificity of the response to a treatment may often be satisfactorily confirmed even when one has to rely on the patient's unsupported statements. But experience shows that this is possible only when neither the patient nor his attendants, nor the final assessor, know when the treatment is or is not in use. This, the so-called "double-blind" technique, is easy to arrange with the aid of dummy preparations when drugs are being tested, but it is scarcely feasible to devise dummy exercises that will satisfy therapists as well as patients. Hence trials dependent on the reports of patients will not meet this need, and some form of objective test must be used. The measurements selected for the trial should obviously include those that the proponents of the treatment hope to influence. If significant improvement in the measurements can be shown to follow a treatment it may be considered vindicated; but a negative result cannot be conclusive, since it is always possible that subjective improvement might depend on factors not accessible to measurement. A situation may thus arise where both subjective and objective assessments prove inconclusive: the former because proper control is impossible, and the latter because its relevance cannot be proved. This situation does appear to obtain in respect to emphysema, where objective tests have yielded consistently negative results, while claims that the patients appear to be relieved continue to be made. In such circumstances I suggest one is fortified by therapeutic history in insisting that the proponent of a time-consuming and expensive treatment should have to prove his case.

At this point I should perhaps state my personal view. It is that exercises are very unlikely to influence the pathological basis of emphysema, and, in particular, the efforts made to achieve more complete chest emptying are based on erroneous concepts. I am therefore disposed to accept the negative results of objective tests as evidence that this class of exercise should be abandoned. I know of only one way in which a patient may be helped by training, and that is by being taught to increase his depth of breathing at the very onset of effort. In this way he anticipates the increased respiratory demand, and during moderate exertion may avoid the development of a respiratory debt which cannot be repaid without resting. Intelligent patients often teach themselves to do this, and the others would probably benefit by instruction and exhortation.

ASTHMA

The foregoing discussion of the difficulty of adequately controlling non-specific effects when relying on patients' reports applies equally in the case of asthma. Moreover, objective measurements of airway resistance, while closely related to disability at the time, cannot usually be taken as representative of long-term trends. Probably the best that can be done is to have the patient keep a diary of attacks; and to arrange as a control period some fairly lengthy treatment requiring a good deal of attention—for example, a desensitization course—adding the exercises when any initial improvement has stabilized.

This modest programme does not appear to have been attempted. If it ever is, I should be surprised if its result confirms the value of exercises.

Asthmatics tend to maintain a large functional residual air even between attacks, and eventually develop a deformity which is essentially a caricature of the inspiratory posture. It seems quite plausible that training designed to encourage full lung emptying might, especially in young patients, check the development of their deformity. But it is one thing to believe such a result might well follow, and another to establish that it does in fact. Only by the comparison over several years of equivalent treated and control groups could the necessary evidence be obtained, and this has not, so far as I know, been attempted.

During an attack a patient may so increase his functional residual air as to abolish his inspiratory reserve, and, feeling that he cannot increase his tidal air, he may become panic-stricken. In fact, he will usually have a considerable expiratory reserve, and the demonstration that a few determined expiratory efforts allow an increase on tidal air often has a most reassuring effect. This procedure, like the encouragement of the emphysematous to anticipate their respiratory needs, is better considered as advice rather than as exercises, and should be the job of the physician rather than the physiotherapist.

CONCLUSION

The conclusion from this survey is that there is no good evidence at present of benefit from breathing exercises; nor is there reason to expect that evidence will be forthcoming, except possibly in the prevention of asthmatic deformity in young subjects.

ORIGINAL PAPER

SOME RESULTS OF REHABILITATION OF INJURED MINERS*

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Durham

THE coalfields of Great Britain are divided by the National Coal Board into nine divisions—eight in England and Wales and one in Scotland. The Durham Division, employing just over 102,000 men, is the second biggest after the South-Western Division (Wales), which employs about the same number. The Durham coalfield occupies the greater part of the county, sparing only the extreme southern and north-western areas. The surface of the county is studded with pit shafts and slag-heaps. There are at present 130 collieries in Durham, though there have been more in the past, and, with the extensive industrial development in the southern part of the county, only its north-western area remains completely unspoilt and beautiful.

The first of the rehabilitation centres for injured miners was started by E. A. Nicoll, at Berry Hill Hall, near Mansfield, in 1939. It was sponsored by a group of local colliery owners and supported by the National Union of Mineworkers. The project proved successful and, wishing to extend such facilities to miners in other areas, the Miners' Welfare Commission established centres in each of the other divisions, except in the small South-Eastern Division.

On the advent of the National Health Service the centres in England and Wales were taken over by the Ministry of Health, each under a separate management committee, but with Mr. Nicoll remaining as consultant adviser. The centre for the Durham Division was established at The Hermitage, Chester-le-Street, in 1944, somewhat appropriately in a former coal-owner's house. It has beds for 60 patients, but unfortunately, because of the length of its drive (just over half a mile), it cannot be satisfactorily used for out-patients. The centre is in the charge of an orthopaedic surgeon working in conjunction with an assistant orthopaedic surgeon and a physician in physical medicine. It is staffed by two or three physiotherapists, one or two remedial gymnasts, and one occupational therapist (technician). The senior physiotherapist is designated chief of treatment staff, and is responsible to the physician in physical medicine for the over-all treatment plan at the centre and the detailed timing of the programme for each individual patient between the various sections.

Any miner who works in the Durham Division and has been injured is eligible for admission. Cases are referred mainly by the hospitals in the area,

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occasionally by general practitioners or the trade unions. In order to qualify for admission a patient must be able to negotiate a flight of stairs. With this condition, therefore, the centre does not in any way relieve the pressure on hospital beds; but, on the other hand, it costs only about £7 a week to keep a miner at the centre, so that, relatively speaking, these are very low-cost beds. Improvements in the turnover have eliminated any waiting list, and consequently patients can usually be admitted at the time most advantageous to them, although it cannot be said that these facilities are always used to the best advantage by some of the hospitals in the area.

In the five years 1952–6 just over 1,500 patients passed through the centre. Their average age was 41 years, and the distribution of injuries was as follows: upper limb 18%, the most frequent of these being to the hand and to the shoulder, in that order; lower limb 50%, the most frequent being torn knee cartilages, fractures of the tibia and of the tibia and fibula, and fractures of the femur, in that order; trunk 30%, the most frequent injuries being to the spine and to the pelvis. Spinal injuries occur frequently from falls of roof and from lifting strains, and pelvic injuries from being crushed by tubs; miscellaneous injuries accounted for 2%. Of the 1,500 patients, about 8% had multiple injuries—that is, a major injury to more than one part of the body.

TABLE I

NUMBERS DISCHARGED FROM THE DURHAM MINERS' REHABILITATION CENTRE, 1952-6

Year	No. of Patients
1952	261
1953	289
1954	291
1955	314
1956	354
Total	1,509

In the period under review the turnover was increased each year—354 patients discharged in 1956 as compared with 261 in 1952 (Table I). This increase in turnover was made possible by a reduction in the average period of stay per patient from 11·7 weeks in 1952 to 9·2 weeks in 1956 (Table II). The factors responsible for this improving turnover were: (1) more thorough evaluation of the patient on admission; (2) more frequent review whilst under treatment; and (3) improvements in the treatment programme. The thorough evaluation of the case on admission is particularly important. Not only is the usual physical and psychological assessment made, but also and especially the

vocational one. The patient's job is considered, the discharge category anticipated, and the prognosis recorded in the notes. In consequence, when the standard of fitness anticipated has been reached the case is considered for discharge. This often saves a number of weeks of needless treatment trying unsuccessfully to obtain full fitness. The importance of this is emphasized by the fact that less than half the patients are able to achieve sufficient fitness to return to full work on discharge from the centre.

Working conditions in the Durham coalfield are arduous: the seams are low, commonly 18 inches to 3 feet, so that for the most part the face worker has to lie on his side or squat on his heels to work and only very rarely can he stand. Hence a very high standard of fitness is required for a man to be able to work a 7½-hour shift in such conditions.

A patient can be discharged in one of six categories: A, full work; B, temporary light work; C, permanent light work; D, unfit for further work in the pits; E, accelerated retirement for a man approaching 65 years of age; and F, requiring further hospital treatment. During the five years in question 40·3% of patients were discharged fit for full work, 37·7% fit for temporary light work, 12·9% fit for permanent light work, and 0·6% unfit for further work in the pits; accelerated retirement was recommended in 0·5%, and 8% returned to hospital for further treatment. Thus 90·9% were discharged fit for some sort of work

TABLE II

AVERAGE PERIOD OF STAY AT REHABILITATION CENTRE

Year	Weeks
1952	11·7
1953	11·1
1954	10·3
1955	9·6
1956	9·2

in the pits. After discharge all patients are followed up for six months, and those in which there is any difficulty in resettlement, until placement has been made. Of those fit for temporary light work, a smaller proportion ultimately became fit for full work than for permanent light work. Thus, comparing the discharge category with the ultimate resettlement category, one finds that the number in category C (permanent light work) has increased from 12·9% to 36·0%, whilst in category A (full work) the number has increased to only 57·0% from 40·3% (Table III). Perhaps the most interesting feature about these results is that in a heavy industry like mining with a paucity of light work,

of the 91·5% patients discharged from the centre fit for work, all but 0·6% (or about one in every 200) are found a job in the pits. For this the main credit is due, not to the medical side, but to the resettlement organization, which is sponsored by the management committee, initiated by the chief clerk at the centre, and carried out by the colliery managers. The management committee, with its strong trade-union element and under the dynamic chairmanship of a prominent miners' leader, is naturally very interested in this problem, and certain of its members, representing personnel and welfare, are specially delegated to look after these interests. When a patient is discharged from the centre with any restriction of category, the chief clerk contacts the colliery manager and gives him details of the limitations imposed (these are specifically mentioned in the doctor's discharge note). The manager then endeavours to get the man suitably placed, giving priority to those who are in category B, or fit for temporary light work, above the ordinary waiting list for light work at the

TABLE III
FINAL RESETTLEMENT CATEGORY AS COMPARED WITH CATEGORY ON DISCHARGE

Category	On Discharge	On Follow-up
	%	%
A	40·3	57·0
B	37·7	—
C	12·9	36·0

colliery. At special quarterly meetings of the management committee, which are attended by the orthopaedic surgeon in charge and the physician in physical medicine, cases not yet resettled are reviewed and further efforts to get them placed planned. The rare case where the patient wants to, or has to, seek work outside the pits is referred to a local hospital resettlement clinic where such exists, or otherwise to the disablement resettlement officer (D.R.O.) in the area where the patient lives. It is in this way that these remarkable results are obtained, although the process is aided by the fact that the miner likes to work amongst his own community, and if he leaves it he will forfeit the free coal and often the free house that go with his job.

The rehabilitation service for injured miners is thus another example of a number of comprehensive projects in Great Britain that cover the needs of certain special sections of the community, others being those established within the R.A.F. and within the motor industry at the Vauxhall and Austin works.

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REVISION SERIES

SYSTEMIC MANIFESTATIONS OF RHEUMATOID ARTHRITIS AND THE PARARHEUMATIC DISEASES*

I—CONCEPT AND PATHOLOGICAL BASIS

By A. T. RICHARDSON

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It is traditional for the opening speaker of a symposium such as this to deal with the general rather than the particular. When the symposium is devoted to the systemic manifestations of rheumatoid arthritis and the pararheumatic diseases this is, perhaps, best achieved by reference to the concept and the pathological basis of these systemic disorders of collagen. In this paper, therefore, these are discussed, and particular reference is made to the validity of the concept.

Although the present grouping together of rheumatic fever, rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, diffuse scleroderma, and dermatomyositis as collagen diseases derives directly from Klinge's studies on rheumatic fever, the idea of systemic disorders of connective tissue had in fact existed previously. For instance, as Robb-Smith (1954) notes in a historical review of the subject, Bichat recognized such a possibility before his early death in 1802. None the less, it was Klinge (1929) who stressed that the essential lesion of rheumatic fever was fibrinoid degeneration of connective tissue, and it was he who identified this with the lesions produced in rabbits rendered hypersensitive to foreign protein. Similarly he (1933, 1934) identified fibrinoid in polyarteritis nodosa and rheumatoid arthritis. This list of diseases was subsequently extended by Masugi and Yä's (1938) demonstration of fibrinoid degeneration in the blood vessels of diffuse scleroderma, which they also interpreted as a hypersensitivity phenomenon. Finally Klemperer, Pollack, and Baehr (1942), in a classic paper, drew attention to the morphological similarities of diffuse scleroderma and acute disseminated lupus erythematosus and introduced the term "diffuse collagen disease". Later these workers also included dermatomyositis, whose similarity to diffuse scleroderma had been emphasized by Oppenheim as long ago as 1903. It is to be noted that Klemperer (1947), to quote his own words, "only wanted to indicate that this tissue [i.e. collagen tissue] may be the common and anatomic site of several diseases", and

* Based on papers read at the Annual Meeting of the British Association of Physical Medicine, April 25, 1958.

he has always denied any aetiological implications of morphological similarities of these disorders.

Alterations of connective tissue in the collagen diseases are, of course, by no means confined to fibrinoid change as described by Klinge and defined by identical eosinophilic staining of ground substance and altered fibres. They consist rather of this with exudation, fibroblastic proliferation, and inflammatory-cell infiltration—changes which by their different degree and distribution allow pathological differentiation of the various constituent disorders. In general, the principal sites of such changes are the joints and related structures in the rheumatic diseases, the blood vessels in lupus erythematosus and polyarteritis, and the skin and fascial planes in scleroderma and dermatomyositis. Additionally, in systemic lupus erythematosus, at least, other distinct lesions exist, as illustrated by haematoxylin bodies and wire-loop lesions of the kidney.

The precise nature of fibrinoid and its relationship to collagen fibres and ground substance still elude us, but from studies on rheumatoid nodules it is possible to define a fibrillary portion comparable to natural collagen and a granular portion which stains for polysaccharides and occasionally for fibrin (Fawns and Landells, 1954). Essentially similar changes are to be found in the nodules of rheumatic fever (Consden, Glynn, and Stanier, 1953). Recent work has also demonstrated that experimentally produced intravascular fibrinoid may be derived from fibrinogen (Pappas, Ross, and Thomas, 1958). In contrast, the haematoxylin bodies first demonstrated in the cardiac lesions in Libman–Sachs disease by Gross (1932), and which are virtually pathognomonic of systemic lupus erythematosus, have their origin from cell nuclei, as indicated by their positive Feulgen reaction. The obtaining of this reaction combined with poor staining by methyl green indicates a content of depolymerized deoxyribose nucleic acid (D.N.A.) (Pollister and Leuchtenberger, 1949), and similar material has also been found in the wire-loops and hyaline bodies of glomeruli (Gueft and Laufer, 1954) and in L.E. cells (Lee, Michael, and Vural, 1951). The obtaining of a positive Feulgen reaction occasionally in extravascular fibrinoid deposits would appear to indicate a component of nuclear material in these also. A further contribution to the structural changes in disseminated lupus erythematosus comes from Mellors, Ortega, and Holman (1957), who have identified serum gamma-globulins in the eosinophilic thickening of the glomerular capillary loops in this disease. It is clear, therefore, that while fibrinoid change as defined by some of its staining characteristics is common to the collagen diseases, its chemical composition is complex and variable in different sites in these diseases.

Klinge's work on associating fibrinoid changes with hypersensitivity was

supported by Clark and Kaplan's (1937) demonstration of this change in serum sickness in man. Later Rich (1942) went so far as to suggest sulphonamides as the allergen in polyarteritis nodosa, and to-day this disease is generally regarded as a hypersensitivity reaction to a bacterial antigen or to drugs, notably to the sulphonamides used in upper respiratory infections (Rose and Spencer, 1957). Rich (1947) also equated the lesions of systemic lupus erythematosus with hypersensitivity. In this way a trend in thinking has developed so that to-day prominent amongst the theories of aetiology and pathogenesis of the collagen diseases are those implicating altered tissue reactivity, disturbances of gamma-globulin metabolism and of antibody formation. Some, indeed, would make an alteration in tissue reactivity the unifying principle of the collagen group of diseases; for example, Selye (1950) refers to diseases of adaptation, Diaz (1951) to dysreaction diseases, and Ehrich (1952) considers the common denominator to be a dysgammaglobulinaemia.

That the changes in the serum globulins are an important factor in this group of diseases is obvious from the references made above to the renal pathology of systemic lupus erythematosus. They are most simply demonstrated by the raised erythrocyte sedimentation rate and raised α_2 and γ globulins which are usual features of these diseases. It is also indicated by the chemical, physical, and serological reactions now in general use in clinical practice, but these also serve to indicate fundamental differences. Thus, as an example of the differentiation of serum globulins in the collagen diseases by their physical properties may be cited the characteristic presence of cryoglobulins in polyarteritis and occasionally in systemic lupus erythematosus; and an example of the chemical differentiation is the obtaining of colloidal gold flocculation in rheumatoid arthritis, but not in rheumatic fever except in the presence of cardiac failure. By serological methods the most specific differentiation of all may occur by the detection of the Rose–Waaler factor and the L.E. factor.

That both the Rose–Waaler factor in rheumatoid arthritis (Glynn, Holborow, and Johnson, 1957) and the serum factor responsible for the L.E.-cell phenomenon (Franklin, Holman, and Muller-Eberhard, 1957) have many of the characteristics of auto-antibodies suggests the production of tissue reactions in these diseases in a way comparable to that of Hashimoto's thyroiditis (Riott *et al.*, 1956) and that of the autohaemolytic anaemias (Dacie, 1954). Additional evidence for the identification of these factors with auto-antibodies comes from Gajdusek's (1958) demonstration of a high incidence of a serum factor in the collagen diseases capable of fixing complement in the presence of tissue extracts. Most interesting of all are the observations by Robbins *et al.* (1957) of absorption of the L.E. factor by cell nucleoprotein, and those of Polli, Celada, and Ceppellini (1957) of the presence of antibody to purified

D.N.A. in a patient with systemic lupus erythematosus. Such results hint strongly at a basic mechanism in this disease—namely, hypersensitivity to D.N.A. proteins. Further, the demonstration of an abnormal globulin with specific reactive affinity for intranuclear D.N.A. in scleroderma, dermatomyositis, and rheumatoid arthritis with L.E. cells, as well as in systemic lupus erythematosus, and the simulation of the characteristics of sera obtained from patients with collagen diseases by that obtained from histone-sensitized rabbits, extends the thesis (Bardawil, Toy, and Galins, 1958).

However, the demonstration of auto-antibodies begs the question of what, if any, is the provocative antigen. Is it an infective agent? Is it a toxic or enzymic change in connective tissue, or is it a combination of a hapten such as a sulphonamide or hydrallazine with connective tissue or nuclear protein, comparable with the "sedormid"–platelet antigen? In this connexion the paper of Glynn and Holborow (1952) on the production of complete antigen capable of producing antibodies to polysaccharide by a combination of serum polysaccharide and β-haemolytic streptococci is noteworthy. Perhaps stimulation of antibody production depends on the degree, place, or repetition of physical contact between antigen- and antibody-producing cells. Again, perhaps the reaction develops because of a primary failure of antibody to recognize normal native tissue; the parallel of haemolytic anaemias in the reticuloses is pertinent to this hypothesis.

Finally, perhaps these diseases are dependent on quantitative changes between antigen and antibody. If so, this is hardly likely to be related to the absolute values of antibody, in view of the demonstration by Good, Rotstein, and Mazzitello (1957) of three cases of rheumatoid arthritis and one of systemic lupus erythematosus with agammaglobulinaemia in a report in which they also refer to Hansen's description of dermatomyositis and van Gelder's of scleroderma with agammaglobulinaemia. In contrast to this, Leonhardt (1957) records a high incidence of systemic lupus erythematosus in familial hypergammaglobulinaemia. At all events, no common aetiology for the collagen diseases has yet been described and the pathogenesis is as yet not clear, although evidence for its dependence on a hypersensitivity mechanism is rapidly accumulating. When they are, of course, they will provide the ultimate justification for the concept of the collagen diseases and a rational classification of them.

In addition to the presence of fibrinoid and the evidence implicating hypersensitivity, an additional important unifying factor for the collagen diseases was the discovery of cortisone. While to-day the application of adrenal steroid therapy in these diseases is limited, undoubtedly it was the discovery by Hench (1949) and subsequent workers that the symptoms of collagen diseases were at

least initially relieved by cortisone that attracted clinicians to what had hitherto been an unattractive morphological concept of doubtful aetiology and without hint of therapy.

Clinically, as well as morphologically and serologically, members of the collagen group of diseases have much in common, in particular the presence of vascular lesions. Difficulties in differential diagnosis must inevitably be many where there is a wide range of clinical manifestations, very few of which are pathognomonic and many of which are common to all. It is little wonder, therefore, that the diagnosis of "vascular collagen disease of unknown type" is sometimes made. In addition, the remarkable variation in the natural history of any one of these diseases often suggests a transition from one type to another —a suggestion that should perhaps be confined to the dissemination of discoid lupus erythematosus. Indefinite ideas such as these do little to assist research into these disorders when it depends so largely on a search for strict diagnostic and prognostic criteria and therapeutic indications. It also confuses teaching and has even led to some popular misconceptions. As an example of this, Dowling (1958) notes the common textbook statement that circumscribed scleroderma or morphoea may develop into generalized disease, the implication being that the viscera may become involved. In clinical reality diffuse scleroderma or progressive systemic sclerosis, a disease in which the viscera are involved and skin changes are characteristically symmetrical but restricted, is quite distinct from morphoea, which, while it may become widely distributed over the skin, does not generally involve the viscera. Reference may also be made to the usual association, in writing, of dermatomyositis and diffuse scleroderma, although visceral lesions such as those found in scleroderma do not occur in dermatomyositis and the association that dermatomyositis has with carcinoma has not been demonstrated for scleroderma. Many more similar examples could be given, all serving to stress the importance of differentiating these diseases both in clinical practice and in teaching. While it must be admitted that differences between members of this group of disorders may be difficult to demonstrate initially, over the course of time and with application of modern diagnostic techniques such as biopsy, serological tests, and even electromyography, an unequivocal diagnosis can usually be made.

In summary, therefore, it may be concluded that the concept of the systemic collagen diseases based originally on morphological characteristics and later on the associated serum globulin changes and implied hypersensitivity mechanism is well founded, although these very changes allow differentiation of the diseases included in this group. Its great value is that, while not implying clinical identity of those diseases, it has stimulated rewarding research and focused attention on systemic diseases of connective tissue and hypersensitivity

mechanisms. Clinical advances *per se*, however, are more likely to come from regarding these diseases as separate entities, and it is in this context that the speakers who follow will be contributing to this symposium.

References

BARDAWIL, W. A., TOY, B. L., and GALINS, N. (1958) *Lancet*, **1**, 888.
CLARK, E., and KAPLAN, B. I. (1937) *Arch. Path.* (*Chicago*), **24**, 458.
CONSDEN, R., GLYNN, L. E., and STANIER, W. N. (1953) *Biochem. J.*, **55**, 248.
DACIE, J. V. (1954) *The Haemolytic Anaemias: Congenital and Acquired.* Churchill, London.
DIAZ, J. C. (1951) *Ann. rheum. Dis.*, **10**, 144.
DOWLING, G. (1958) *Med. Press*, **239**, 92.
EHRICH, W. E. (1952) *Amer. Heart J.*, **43**, 121.
FAWNS, H. T., and LANDELLS, J. W. (1954) *Ann. rheum. Dis.*, **13**, 28.
FRANKLIN, E. C., HOLMAN, M. R., and MULLER-EBERHARD, H. J. (1957) *J. exp. Med.*, **105**, 425.
GAJDUSEK, D. C. (1958) *A.M.A. Arch. intern. Med.*, **101**, 9.
GLYNN, L. E., and HOLBOROW, E. J. (1952) *Lancet*, **2**, 449.
——— ——— and JOHNSON, G. D. (1957) *Proc. roy. Soc. Med.*, **50**, 469.
GOOD, R. A., ROTSTEIN, J., and MAZZITELLO, W. F. (1957) *Ann. rheum. Dis.*, **16**, 131.
GROSS, L. (1932) *Contributions to the Medical Sciences in Honour of Emanuel Libman*, **2**, 527. International Press, New York.
GUEFT, B., and LAUFER, A. (1954) *A.M.A. Arch. Path.*, **27**, 201.
HENCH, P. S. (1949) *Proc. Mayo Clin.*, **24**, 181.
KLEMPERER, P. (1947) *Bull. N.Y. Acad. Med.*, **23**, 581.
——— POLLACK, A. D., and BAEHR, G. (1942) *J. Amer. med. Ass.*, **119**, 331.
KLINGE, F. (1929) *Beitr. path. Anat.*, **83**, 186.
——— (1933) *Ergebn. allg. Path. path. Anat.*, **27**, 1.
——— (1934) *Handbuch der speziellen pathologischen Anatomie und Histologie*, ed. F. Heuke and O. Lubarsch, **9/2**, 107.
LEE, S. L., MICHAEL, S. R., and VURAL, I. L. (1951) *Amer. J. Med.*, **10**, 446.
LEONHARDT, T. (1957) *Lancet*, **2**, 1200.
MASUGI, M., and YÄ, S. (1938) *Virchows Arch. path. Anat.*, **302**, 39.
MELLORS, R. C., ORTEGA, L. G., and HOLMAN, H. R. (1957) *J. exp. Med.*, **106**, 191.
PAPPAS, G. D., ROSS, M. H., and THOMAS, L. (1958) Ibid., **107**, 333.
POLLI, E., CELADA, F., and CEPPELLINI, R. A. (1957) *Boll. Ist. sieroter. milan.*, **36**, 454.
POLLISTER, A. W., and LEUCHTENBERGER, C. (1949) *Proc. nat. Acad. Sci.* (*Wash.*), **35**, 111.
RICH, A. R. (1942) *Bull. Johns Hopk. Hosp.*, **71**, 123.
——— (1947) *Harvey Lect.*, **42**, 106.
RIOTT, I. M., DONIACH, D., CAMPBELL, P. M., and HUDSON, R. V. (1956) *Lancet*, **2**, 820.
ROBBINS, W. C., HOLMAN, H. R., DEICHER, H., and KUNKEL, H. G. (1957) *Proc. Soc. exp. Biol.* (*N.Y.*), **96**, 575.
ROBB-SMITH, A. H. T. (1954) *Practitioner*, **173**, 117.
ROSE, G. A., and SPENCER, H. (1957) *Quart. J. Med.*, **26**, 43.
SELYE, H. (1950) *The Physiology and Pathology of Exposure to Stress.* Acta Inc., Montreal.

II—RHEUMATOID ARTHRITIS

By R. M. MASON and V. L. STEINBERG
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THE systemic manifestations of rheumatoid arthritis are many and various (Table I), but we shall describe only those which seemed to us to be of both practical and theoretical importance, of topical interest, and of which we have had personal experience in the past few years. These are three: (1) pulmonary (including pleural) changes; (2) peripheral neuropathy; and (3) arteritis. For purposes of description, at least, we have kept them separate.

TABLE I
SYSTEMIC MANIFESTATIONS OF RHEUMATOID ARTHRITIS

I. Pyrexia
II. Nodules
III. Arteritis
IV. Pancarditis
V. Peripheral neuropathy
VI. Respiratory:
  Pleurisy
  Lung parenchymatous changes
VII. Reticulo-endothelial:
  Lymphadenopathy
  Splenomegaly
  Hepatomegaly
VIII. Haematological: (Felty's syndrome)
  Anaemia
  Leucocytosis
  Leucopenia
IX. Ocular:
  Uveitis
  Sjögren's syndrome
  Band-shaped opacities
  Scleromalacia perforans
  Episcleritis
X. Skin:
  Sweating
  Rash
  Chronic ulcers
XI. Amyloidosis

1. PULMONARY MANIFESTATIONS

We have observed 15 patients in which the question arose at some time whether they were representative of the so-called rheumatoid lung, first described by Ellman and Ball (1948). These cases may be classified as follows:

Group I	Pleural effusion without lung changes	6 cases
Group II	Diffuse lung parenchymatous change of known cause	4 cases
Group III	Diffuse lung parenchymatous change of undetermined cause (possible rheumatoid lung)	4 cases
Group IV	Systemic lupus erythematosus	1 case

GROUP I: PLEURAL EFFUSION WITHOUT LUNG CHANGES

In the six cases in this group (Table II) no evidence of tuberculosis or neoplasm has been found either at the time or at follow-up. There is nothing exceptional about the age of the patient or the duration of disease in these cases, which are strictly comparable with our material as a whole. It is, however,

remarkable that all were in males, in contrast to the 2 : 1 ratio of females to males in our whole series. There does appear to be an association between the pleural effusion and the arthritis to the extent that in every case there was either a concomitant flare of joint symptoms or, in one case (Case 5), rapidly progressive joint disease associated with arteritic phenomena. This case is referred to below and is the only one in which arteritis has been diagnosed clinically at a later date. Emerson (1956) has also described six very similar cases; in these there was also a flare of the arthritis in association with the effusion. Only three of our cases had subcutaneous nodules. It would seem reasonable to suppose that where nodules are a marked feature of the disease,

TABLE II

PLEURAL EFFUSION AND RHEUMATOID ARTHRITIS (GROUP I)

Case No.	Sex and Age		Duration of Arthritis (Years)	Association with Joint Flare	D.A.T.	L.E. Cells	Subcutaneous Nodules
1	M	46	5	+	256	—	—
2	M	39	10	+	32	—	—
3	M	59	11	+	256	—	++
4	M	51	2	+	2056	—	+++
5	M	62	3	+	64	—	+
6	M	46	3/12	+	16	—	—

Mean age 50·5 years. Mean duration of arthritis 5·2 years.

pleural nodules, which have been described in autopsy material (Raven *et al.*, 1948; Ellman *et al.*, 1954) might be responsible. One case (Case 4; Plate IV, Fig. 1) had multiple nodules as well as a pleural effusion. The effusion has cleared, however, and the patient is back at work in partial remission without steroid administration. It does not seem to us likely that rheumatoid nodules can be the only explanation of these effusions. Coincidental tuberculosis is, of course, a real possibility; in Case 1 this was strongly suspected at one time. An encysted basal effusion was present (Plate IV, Fig. 2), and this was still visible 21 months later, but after four years no more than residual pleural thickening was found. Tubercle bacilli were never demonstrated.

We thus have a curious group of rheumatoid arthritics presenting a fairly consistent pattern, all male, all with a pleural effusion for which no confirmation of a tuberculous or neoplastic aetiology was found, all having a flare of joint symptoms associated with the effusion, all clearing up without specific therapy in the course of months or years, and only one developed other arteritic phenomena. Only three of the six had clinical rheumatoid nodules.

Group II: Diffuse Lung Parenchymatous Change of Known Cause

This group comprises four cases in which diffuse pulmonary changes were observed which might on superficial study have been regarded as a "rheumatoid lung" of the diffuse type (Ellman, 1956), but for which we believe we have a convincing alternative explanation.

Case 7.—Male aged 58 with a 14-year history of chronic severe rheumatoid arthritis. Sheep-cell differential agglutination titre (D.A.T.) 1 in 64. He complained of dyspnoea, but there were no abnormal physical signs in the chest. He had been a stonemason for many years, and this led to a diagnosis of silicosis.

Case 8.—Male aged 58 with rheumatoid arthritis of eight years' duration. D.A.T. 1 in 32. Three years previously he had had a pleural effusion. A radiograph showed fibrosis in both lower zones. This was puzzling until we found that he applied paraffin nasal drops every night on going to bed. He was seen by Dr. Rusby, who considered that he had oil lipoidosis.

Case 9.—Male aged 44 with rheumatoid arthritis of 20 years' duration. Radiography showed diffuse mottling. He had been a tool-grinder for 25 years, and this was considered to be responsible for the condition of the lung.

Case 10.—Female aged 47 with rheumatoid arthritis of three years' duration. D.A.T. 1 in 32. Complained of cough and sputum. Radiograph showed a characteristic ground-glass appearance. The fact that she had worked with asbestos 25 years previously suggested a diagnosis of asbestosis.

The four cases in Group II are summarized in Table III.

TABLE III

Diffuse Lung Parenchymatous Change of Known Cause in Rheumatoid Arthritis (Group II)

Case No.	Sex and Age	Duration of Arthritis (Years)	Diagnosis	Cause
7	M 58	14	Silicosis	Occupational (stonemason)
8	M 58	8	Oil lipoidosis	Paraffin drops
9	M 44	20	Trade lung	Occupational (tool-grinder)
10	F 47	3	Asbestosis	Occupational (asbestos trade)

Group III: Diffuse Lung Parenchymatous Changes of Undetermined Cause

There remain, however, a residue of four cases in which diffuse lung changes were found for which we could find no cause, and we cannot therefore exclude the possibility that these represented rheumatoid lungs.

CASE 11.—Male aged 61. In 1946 he attended the Orthopaedic Department at the London Hospital, when clubbing of the fingers and toes was noted (Plate IV, Fig. 3). Chest radiograph showed diffuse reticulation (Plate V, Fig. 1). In 1951 he developed rheumatoid arthritis, typical clinically, radiologically, serologically, and histologically. There is marked impairment of pulmonary function tests.

CASE 12.—Female aged 55 with rheumatoid arthritis of two years' duration. D.A.T. positive. L.E. cell negative. No chest symptoms. Chest radiograph showed diffuse mottling.

CASE 13.—Female aged 52 with rheumatoid arthritis of five years' duration. D.A.T. 1 in 32. L.E. cell negative. Chest radiograph showed increased mediastinal shadowing and pulmonary fibrosis (Plate V, Fig. 2). Treated with prednisone without any change in appearance of chest radiograph.

CASE 14.—Male aged 46 with rheumatoid arthritis of nine months' duration. D.A.T. 1 in 256. Radiograph of chest, which was asymptomatic, showed diffuse reticulation.

It must be confessed that these four cases do not present any very striking pattern, and the diagnosis rests largely on an inability to find any other cause for these appearances. We have some sympathy with Aronoff and his colleagues (1955), who from a study of 253 cases of rheumatoid arthritis reported that the incidence of pulmonary disease "did not impress them as very different from what might be expected in a general hospital population". They were in diagnostic difficulty in only four cases. The diagnosis in these was considered to be neoplasm, xanthomatosis, sarcoid or xanthomatosis, and "idiopathic pulmonary fibrosis" respectively. Nevertheless Price and Skelton (1956) have shown that arterial changes in the lungs can occur in this disease, and in view of the very real significance of arteritis in rheumatoid arthritis we cannot dismiss the possibility that these lesions are manifestations of arteritis. On the other hand, none of these cases have shown other arteritic phenomena.

GROUP IV: SYSTEMIC LUPUS ERYTHEMATOSUS

We have included one case of lupus erythematosus for comparison. This (Case 15) was referred to us as a case of rheumatoid arthritis, but the clinical diagnosis was systemic lupus erythematosus, and large numbers of L.E. cells were in fact found in the blood. The patient subsequently died. Her lung picture showed diffuse mottling, changes similar to those already described by Rapaport *et al.* (1953) and others.

2. PERIPHERAL NEUROPATHY

It is a curious fact that the modern textbooks on rheumatic disease, both British and American, make no mention of this complication of rheumatoid

FIG. 1

FIG. 2

FIG. 3

FIG. 1.—Case 4. Pleural effusion without lung changes. Showing nodules on elbows.

FIG. 2.—Case 1. Pleural effusion without lung changes. Radiograph showing encysted basal effusion.

FIG. 3.—Case 11. Hypertrophic pulmonary osteoarthropathy and rheumatoid arthritis. Showing clubbing of fingers.

M. & S. facing page 268]

PLATE V

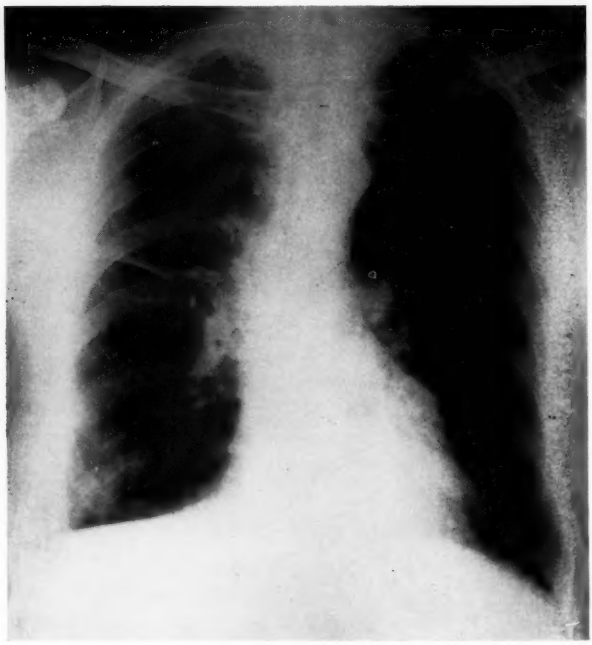

FIG. 1.—Case 11. Hypertrophic pulmonary osteoarthropathy and rheumatoid arthritis. Diffuse pulmonary fibrosis of undetermined cause.

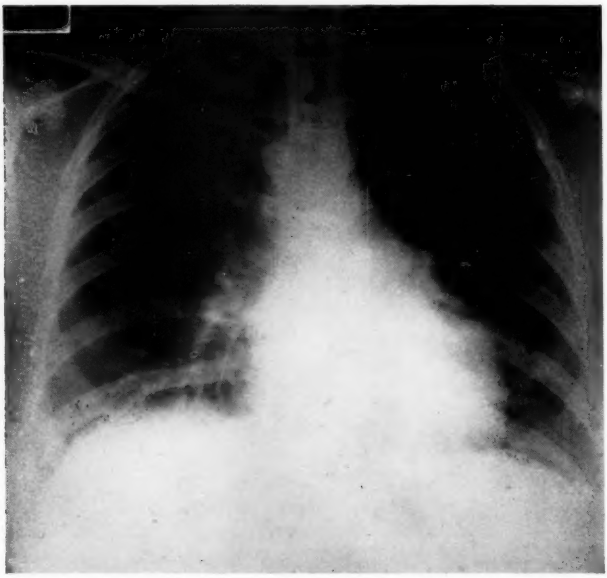

FIG. 2.—Case 13. Pulmonary fibrosis and increased mediastinal shadowing in a case of rheumatoid arthritis.

arthritis. In one book the only mention is in the chapter on hydrotherapy and spa treatment, where amongst details of the technique of administering a mud pack it is remarked that mud is especially valuable in some cases of rheumatic neuritis. Hart and his colleagues (1957) recently reported eleven cases (two of these were patients of Dr. W. S. Tegner), ten with rheumatoid arthritis and one with systemic lupus erythematosus. We have observed six cases so far; five are of definite rheumatoid arthritis by American Rheumatism Association criteria, and one probably rheumatoid arthritis, although we suspect the eventual diagnosis will be lupus erythematosus. Table IV gives brief clinical data on

TABLE IV

CLINICAL DATA ON SIX CASES OF RHEUMATOID ARTHRITIS WITH PERIPHERAL NEUROPATHY

Case No.	Sex and Age	Duration of Arthritis (Years)	D.A.T.	Course of Rheumatoid Arthritis
16	F 68	2	−(×4)	Fluctuating
17	F 64	6	0	Died 9 months after onset
18	M 71	2	+	Died 1 month after onset
19	F 64	1	+	Died 2 weeks after onset
20	M 52	6	0	Progressive
21	M 54	4	+	Progressive
Hart *et al.* (1957)	6 M, 4 F (Mean) 61	6 (Mean)	8+ 20	No relation to flare of rheumatoid arthritis; 1 died

these patients; we have included Hart's series for comparison. In both series there is again a relative preponderance of males over females. The duration of disease in our series is less than that of Hart's. It seems that the severity of the arthritis was greater, for three of our patients died, nine months, one month, and two weeks after the onset of neuropathy, and the prognosis for at least one of the others (Case 21) is very doubtful at this time. The neuropathy itself is commonly sensory, although motor changes may follow.

Two only of our six cases developed motor weakness (Table V). Both these patients died, although one had recovered motor power before death. The lower limb was affected in all but one (Case 20), and he had arteritic changes affecting the same hand. The neuropathy presented with paraesthesiae and numbness; motor weakness, when it occurred, developed later.

A point that must be considered is whether steroid therapy has any relation-

ship to this phenomenon. The steroid history in these cases is shown in Table VI. In no case was there a close relationship to withdrawal of steroid treatment, but in two patients withdrawal or interruption preceded the development of neuro-

TABLE V

CLINICAL DATA ON SIX CASES OF RHEUMATOID ARTHRITIS WITH PERIPHERAL NEUROPATHY

Case No.	Motor Symptoms	Sensory Symptoms	Course of Neuropathy
16	0	+	Static 3 months
17	+	+	Recovered before death
18	0	+	Static 1 month (died)
19	+	+	Died within 2 weeks
20	0	+	Static 1 month
21	0	+	Improving
Total	2	6	
Hart *et al.* (1957)	5	10	3 recovered; 3 improved; 3 static; 1 died

TABLE VI

RELATIONSHIP OF STEROID ADMINISTRATION TO NEUROPATHY IN SIX CASES OF RHEUMATOID ARTHRITIS

Case No.	Onset of Neuropathy
16	3 months after 1 year on prednisone 15 mg. per day
17	Cortisone 37·5–50 mg. per day for 4 years. On prednisolone 10–15 mg. per day for 1 year
18	Prednisolone 15 mg. per day for 1 year. Interrupted abruptly (surgeon) 3 months before onset
19	ACTH 40 units per day for 7 days
20	Prednisone 15 mg. per day for 1 year
21	Prednisone 15 mg. per day for 1 year
Hart *et al.* (1957)	3 related to withdrawal; 7 no relation (7 out of 10 cases on steroids)

pathy by three months. That all these cases were on steroid therapy should, in our view, at present be regarded as an indication of the severity of the rheumatoid process rather than as the cause of the neuropathy.

Other features of these cases which may be of interest are shown in Table VII. Thus three of these patients had other manifestations of arteritis, which

TABLE VII

ASSOCIATED FEATURES IN SIX CASES OF RHEUMATOID ARTHRITIS WITH PERIPHERAL NEUROPATHY

Case No.	Associated Features
16	Splenomegaly, hepatomegaly, leucopenia, albuminuria. D.A.T. negative × 4. L.E. cells not found. Suspect D.L.E.
17	P.U.O. prior to onset of neuropathy
18	Gangrene of toe requiring amputation; arteritis
19	Renal arteritis
20	Digital arteritis
21	Malabsorption syndrome following gastrectomy

would be consistent with the evidence suggesting that the neuropathy of rheumatoid arthritis is due to ischaemic changes secondary to arteritis.

3. ARTERITIS

It is only within the last five years that the importance of arteritis in rheumatoid arthritis has come to be recognized. Slocumb (1953) first drew attention to the "panangiitic reaction" as an effect of cortisone withdrawal in rheumatoid arthritis, although Dawson (1940) had mentioned polyarteritis nodosa in association with rheumatoid arthritis and Sokoloff *et al.* (1951) had found arteritis in occasional muscle biopsies in rheumatoid arthritis. Slocumb, indeed, mentioned peripheral neuropathy as a manifestation of this panangiitis. Cruickshank (1954) reported on 72 autopsies performed on cases of rheumatoid arthritis, of which he found evidence of arteritis in 18, or 25%. Of these 18 cases nine had involvement of the myocardium, five of nerve, one of synovium, three of muscle, and three elsewhere, affecting the small arteries of muscular type, occasionally with necrosis but without aneurysm formation. In the same year Ball (1954) described four cases of rheumatoid arthritis and one case of polyarteritis nodosa with a rheumatoid type of arthritis in which changes indistinguishable from polyarteritis nodosa were found. These included, he had noted, two with pleural effusions and one with peripheral neuropathy. In no case were the changes related to steroid therapy, only one of the four patients with rheumatoid arthritis having had such treatment. In three cases the polyarteritic phenomena were terminal. We have observed presumptive or definite arteritic phenomena in five cases. Some of these have already been mentioned, since they have either pulmonary or neuropathic manifestations for which the arteritis may also have been responsible.

CASE 20.—Male aged 52 suffering from progressive rheumatoid arthritis of six years' duration with multiple subcutaneous nodules. His brother also has severe rheumatoid arthritis. He was taking 15 mg. of prednisone daily. When he woke up one morning during a holiday abroad the fourth and fifth finger-tips of one hand were cyanotic and intensely painful (Plate VI, Fig. 1). Small necrotic spots were also noted on the pulp and around the nail beds (Plate VI, Fig. 2)—appearances identical with those so well illustrated by Bywaters (1957). Full in-patient investigation revealed no other evidence of diffuse vascular change, but six months later he had begun to lose some sensation in his finger-tips and there was slight impairment of appreciation of pinprick in the tips of the fingers of the same hand. This was clearly an arteritic peripheral neuropathy of sensory type, and many more small necrotic foci in the hand developed.

CASE 18.—Male aged 71 with severe fulminating rheumatoid arthritis with progressive deterioration despite treatment with prednisolone, 15 mg. daily. He developed frank gangrene of one fifth toe which had led to his admission to a local hospital, where the prednisolone was abruptly stopped and the toe amputated. He was subsequently readmitted to the London Hospital with florid rheumatoid arthritis, and despite further prednisolone went rapidly downhill, developing a peripheral sensory neuropathy and dying of heart failure attributed to coronary occlusion. No autopsy was carried out.

CASE 5.—Male aged 62 with progressive rheumatoid arthritis of three years' duration and pleural effusion who developed small necrotic foci in the feet.

CASE 22.—Female aged 42 with rheumatoid arthritis of 3½ years' duration. D.A.T. positive. Admitted with acutely painful left big toe and left fourth toe attributed to arteritis.

CASE 19.—Female aged 64. Peripheral neuropathy developed just before her death from bronchopneumonia and pyaemia. Autopsy showed aneurysmal dilatation of two arcuate vessels in the left kidney suggestive of polyarteritis nodosa, and histological examination revealed arteritis in the kidney, nerve, and myocardium (Plate VII).

Thus of our five patients two died, and the three still alive are not very secure. The association between arteritis and steroid administration clearly still deserves serious consideration. Kemper, Baggenstoss, and Slocumb (1957) likewise gained the impression that arteritis in the course of rheumatoid arthritis had become more common during the past few years, and they therefore reviewed all cases of rheumatoid arthritis which came to autopsy at the Mayo Clinic during 1954. They had 52 cases, of which 14 had received cortisone and 38 no steroid at all. They found evidence of "vasculitis" in 12 cases (23%). Of these, in five cases it was limited to the heart and four had evidence of rheumatic fever; none had had cortisone. Three cases had an acute but very localized vasculitis, but none of them had had steroids. Four, or 8%, of the 52 cases showed changes of classical polyarteritis nodosa with diffuse necrotizing

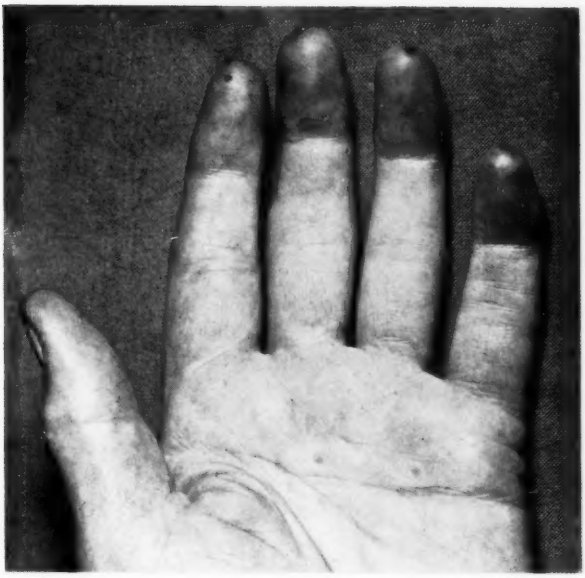

FIG. 1.—Case 20. Digital arteritis in rheumatoid arthritis. Showing cyanosis of finger-tips and localized necrotic areas.

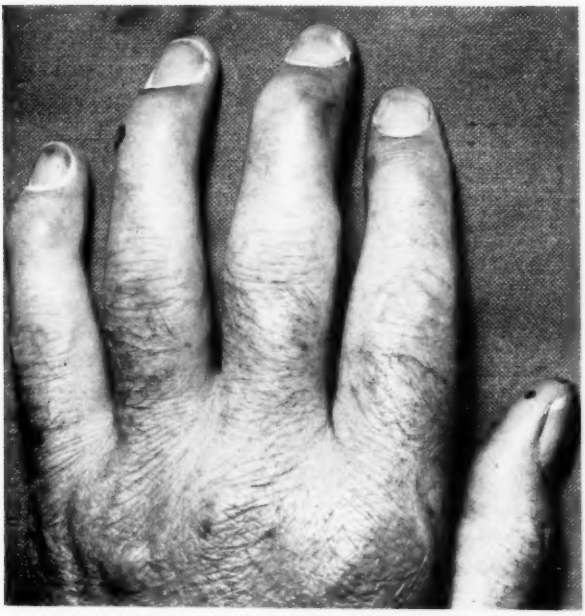

FIG. 2.—Case 20. Digital arteritis in rheumatoid arthritis. Showing localized necrotic areas.

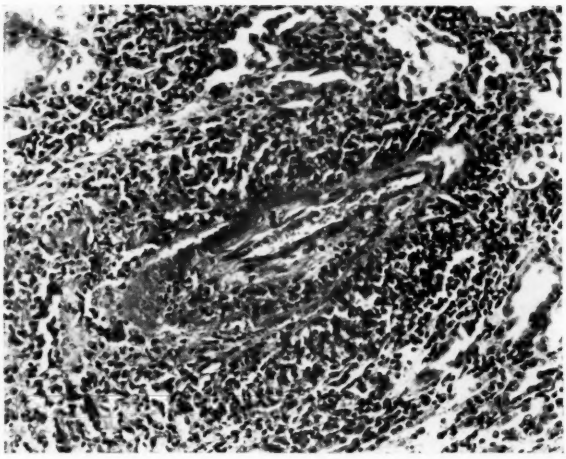

FIG. 1.—Case 19. Section of kidney showing arteritis.

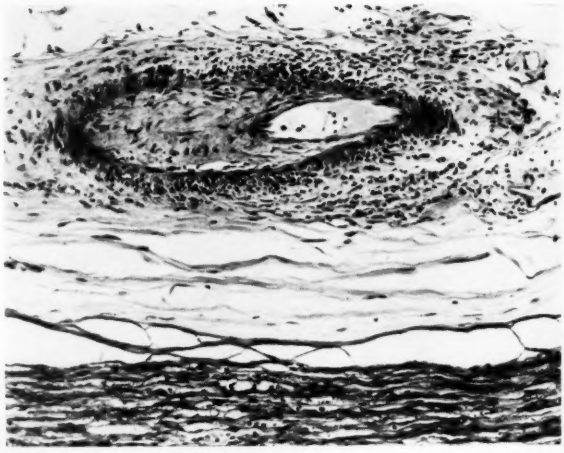

FIG. 2.—Case 19. Section of nerve showing arteritis.

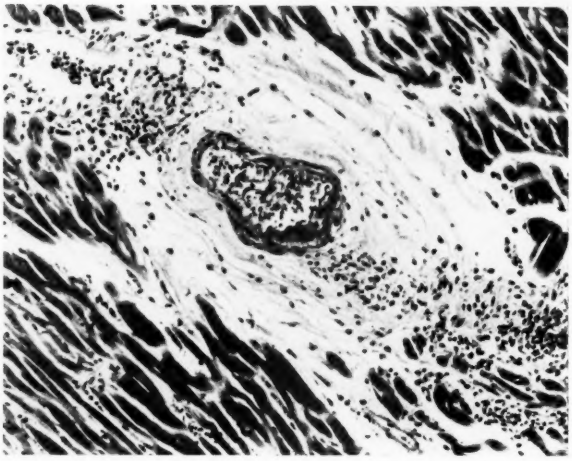

FIG. 3.—Case 19. Section of myocardium showing arteritis.

arteritis affecting most tissues except (and this is curious) the lungs. All had received cortisone. Three of these patients had had a peripheral neuropathy during life.

CONCLUSION

It seems clear that arteritis is a relatively infrequent, although serious, complication of rheumatoid arthritis. Sokoloff and Bunim (1957) report ten cases only seen during four years at the National Institute of Health, Bethesda, Maryland. These include four with pleural effusions and three with peripheral neuropathy. In the light of these reports and of our own experience, therefore, it would seem reasonable to conclude that, in addition to the frank arteritis of superficial tissues, peripheral neuropathy and possibly pleural involvement are manifestations of a single process—arteritis. Whether this is associated with steroid therapy, or simply with severe rheumatoid arthritis which is likely to be treated by steroids, we have insufficient data to dogmatize at present. It does seem likely that sudden withdrawal of steroids may be a dangerous procedure, from this as well as from other aspects, and may set off a diffuse arteritic reaction sometimes necrotizing, which can be manifested in one of several ways.

ACKNOWLEDGMENTS

We would like to thank Dr. W. S. Tegner for permission to report cases under his care, and Dr. J. W. Landells for preparing the sections.

REFERENCES

ARONOFF, A., BYWATERS, E. G. L., and FEARNLEY, G. R. (1955) *Brit. med. J.*, **2**, 228.
BALL, J. (1954) *Ann. rheum. Dis.*, **13**, 277.
BYWATERS, E. G. L. (1957) Ibid., **16**, 84.
CRUICKSHANK, B. (1954) Ibid., **13**, 136.
DAWSON, N. H. (1940) *Ann. intern. Med.*, **13**, 1837.
ELLMAN, P. (1956) *Post-Grad. med. J.*, **32**, 370.
——— and BALL, R. E. (1948) *Brit. med. J.*, **2**, 816.
——— CUDKOWICZ, L., and ELWOOD, J. S. (1954) *J. clin. Path.*, **7**, 239.
EMERSON, P. A. (1956) *Brit. med. J.*, **1**, 428.
HART, F. D., GOLDING, J. R., and MACKENZIE, D. H. (1957). *Ann. rheum. Dis.*, **16**, 471.
KEMPER, J. W., BAGGENSTOSS, A. H., and SLOCUMB, C. H. (1957) *Ann. intern. Med.*, **46**, 831.
PRICE, T. M. L., and SKELTON, M. O. (1956) *Thorax*, **11**, 234.
RAPAPORT, S. I., MEISTER, L., STEELE, F. M., and CANIGLIA, S. R. (1953) *Ann. rheum. Dis.*, **12**, 268.
RAVEN, R. W., WEBER, F. P., and PRICE, L. W. (1948) Ibid., **7**, 63.
SLOCUMB, C. H. (1953) *Proc. Mayo Clin.*, **28**, 655.
SOKOLOFF, L., and BUNIM, J. J. (1957) *J. chron. Dis.*, **5**, 668.
——— WILENS, S. L., and BUNIM, J. J. (1951) *Amer. J. Path.*, **27**, 157.

(Papers III and IV will be published in the November issue)

THE FIFTEENTH ANNUAL MEETING

THE fifteenth annual meeting of the Association was held at the Royal Free Hospital, London, on April 25 and 26, 1958, with the President, Dr. P. Bauwens, in the Chair.

SCIENTIFIC SESSIONS

SYMPOSIUM ON THE SYSTEMIC MANIFESTATIONS OF RHEUMATOID ARTHRITIS AND THE PARARHEUMATIC DISEASES

(April 25: afternoon)

The four opening speakers were DR. A. T. RICHARDSON, DR. R. M. MASON, DR. A. C. BOYLE, and DR. S. MATTINGLY, and taken together their papers comprised a comprehensive review of the so-called collagen diseases. (The first two papers appear in this issue—see page 259.) The general discussion which followed was confined mainly to the elaboration of specific points raised by the opening speakers.

SHORT PAPERS

(April 26: afternoon)

These papers will be published in the *Annals* at a later date, but the following abstracts are given for the benefit of readers who were unable to be present at the meeting.

"*A Comparative Study of Electrodiagnostic and Electromyographic Techniques*", by DR. L. AMICK (co-author DR. R. P. HICKEY).

A comparison was made of the value of progressive current ratios, intensity duration ratios, and constant-current and constant-voltage intensity duration curves in the detection of muscle denervation following peripheral nerve injuries. Except in the case of polymyositis, when constant-voltage stimulators were found to be more reliable, constant-current and constant-voltage stimulators were equally effective in all conditions. Progressive-current stimulators were of doubtful value. Comparison of the constant-current and the constant-voltage stimulator with the electromyograph showed that the latter was more reliable for the detection of minimal denervation. It was felt that the efficiency of the electromyograph could be improved still further by more extensive muscle sampling than was used in the present study. Since no one method was infallible, it was considered that the constant-current or constant-voltage stimulator and the electromyograph should be regarded as complementary in every case.

"*A Preliminary Report on the Assessment of Mucoprotein Levels and Other Serological Reactions in Rheumatoid Arthritis*", by DR. C. FELDMAN (co-authors DR. E. V. HESS and MR. R. L. MARKHAM).

Twenty-three patients suffering from rheumatoid arthritis had been assessed clinically at monthly intervals, and at the same time estimations made of the E.S.R., C-reactive protein, sheep-cell agglutination reaction, and serum mucoproteins. Of these tests the serum mucoprotein concentration showed the most consistent correlation with changes in the clinical state in individual patients, but caution was needed in assessing the results of single as opposed to serial readings.

"*Laboratory Indices in Rheumatic Diseases*", by DR. J. H. GLYN.

In 330 patients suffering from various rheumatic disorders estimations had been made of the E.S.R., and also of C-reactive protein, total protein-bound polysaccharides, total protein, and protein : polysaccharides ratio. As a measurement of disease activity these tests were approximately as accurate as the E.S.R., and the Winzler mucoproteins were found to be the most reliable. All the tests were unreliable when applied to those diagnostic problems in which the clinician most required help, and there was no justification for the routine performance of such time-consuming procedures.

"*The Use of a Frequency Analyser in Clinical Electrodiagnosis*", by DR. R. P. HICKEY.

After reviewing the theory of automatic frequency analysis of the electromyographic interference pattern, Dr. Hickey went on to describe a method of measuring the ratio of low- and high-frequency output which indicated variations of each component and allowed calibration and monitoring. There were limitations to the usefulness of this method and the apparatus was rather elaborate, though further experience might enable it to be simplified. Changes in the frequency ratio with varying contractions and with different positions of the needle electrode could be observed directly without delay, and a good indication was obtained as to which frequency component was varying. By examination of the limb muscles myopathy and polymyositis could be distinguished fairly simply, but peripheral neuropathy and motor neurone disease could easily be confused, and a diagnosis should therefore always depend on a complete electrodiagnostic investigation.

"*Therapeutic Trials in Lumbar Disk Degeneration*", by DR. J. STEWART LAWRENCE.

This paper reported an investigation into the treatment of miners complaining of pain of lumbar or sciatic distribution associated with radiological evidence of disk degeneration but without neurological signs. Immobilization of the lumbar spine by either a plaster jacket or bed rest was found to delay recovery. Mobilization, on the other hand, produced a significantly better result, and manipulation by a physiotherapist was more effective than ionization. However, active mobilization of the spine by exercises was more effective than manipulation. Massage produced temporary relief of pain, although it appeared to have no influence on the outcome of the malady and did nothing to hasten return to work. Cortisone given by mouth to patients in the acute stage of their disability produced no significant difference when compared with similar cases treated by a suspension of cholesterol. The value of a light spinal support to heavy manual workers as a preventive of future painful and disabling attacks of pain remained unproven.

"*Experiences with an Experimental Spinal Brace*", by SQUADRON-LEADER J. L. MILLIGAN.

During the past year clinical trials had been made of a spinal brace based on an original design by Norton and Brown, of Boston, Massachusetts. It consisted of two horizontal bars, one at or just below the level of the posterior iliac spines, and the other at the level of the 12th thoracic vertebra. The bars were connected by lateral members in the mid-axillary line and extended downwards to the level of the greater trochanter of the femur, at which site they terminated in a padded disk. The brace was

secured by a chest strap to the upper bar and by three straps from the lateral members to the central abdominal pad. Extreme simplicity was one of the great advantages of this apparatus, which had been made by technical tradesmen as part of their treatment at a Royal Air Force rehabilitation unit. It was comfortable to wear and produced good immobilization in all positions, even when the subject was sitting and attempting full forward flexion. The spinal extensor muscles could be actively contracted within the brace and their normal use was not prevented.

"*Injuries to the Collateral Ligaments of the Knee-joint*", by FLIGHT-LIEUTENANT V. B. WHITTAKER.

Dr. Whittaker described the results of treatment in 83 cases of collateral ligament injury. No case with associated meniscus or bony injury was included. The ratio of medial to lateral ligament injuries in this series was 7 : 1, and, as in the case of meniscus injuries, Association football was responsible for the large majority of cases. Treatment by complete immobilization, with or without operative repair, produced a greater number of successful results (89%) than treatment by other less radical methods (50%), such as heat therapy, strapping, manipulation, rest in a back splint, or no treatment at all. Immobilization in plaster of Paris for a period of five to six weeks was necessary to achieve a good result, the two cases in this series recorded as failures having been immobilized for less than three weeks.

CLINICAL MEETING

(April 25: morning)

The following cases were presented:

DR. A. T. RICHARDSON:	Dermatomyositis associated with carcinoma treated by hypophysectomy
	Charcot–Marie–Tooth disease localized to the arms
	Unilateral hypertrophy of the calf muscles
	Lead palsy
DR. R. P. HICKEY:	Post-encephalitic rigidity ("stiff man" syndrome?)
DR. E. A. KAUFFMANN:	Ankylosing spondylitis with amyloid disease
DR. C. FELDMAN:	Rheumatoid arthritis with generalized lymphadenopathy
DR. N. A BEARDWELL:	Chronic tophaceous gout with involvement of the sacro-iliac joints
DR. E. V. HESS:	Systemic lupus erythematosus treated with chloroquine
	Systemic lupus erythematosus and pulmonary tuberculosis treated with prednisolone

DEMONSTRATIONS

(April 26: morning)

The demonstrations were arranged specially to appeal to a wide range of interests and included pathological specimens and slides, radiographs, and electrodiagnostic

techniques. Particular interest was shown in the electromyographic frequency analyser and in the demonstration of the use of the serum mucoproteins, C-reactive protein, and agglutination reactions in the diagnosis of the collagen diseases.

At the conclusion of the Annual Meeting the President expressed the deep appreciation the Members of the Association felt for the way in which Dr. Richardson and his staff and others at the Royal Free Hospital had organized a highly successful programme combined with such excellent hospitality.

Annual Dinner

The annual dinner of the Association was held at the Royal College of Physicians on April 25. Distinguished guests included Professor Robert Platt, President of the Royal College of Physicians; Sir Cecil Wakeley, President of the Chartered Society of Physiotherapy; Lord Amulree, President of the Association of Occupational Therapists; Professor A. M. Claye, President of the Royal College of Obstetricians and Gynaecologists; Sir John Charles, Chief Medical Officer, Ministry of Health; Dr. Ian Grant, President of the College of General Practitioners; Miss M. R. Smythe, Chairman of the Council of the Association of Occupational Therapists; Dr. I. Douglas-Wilson, Deputy Editor of the *Lancet*; and Dr. J. G. Thwaites, Assistant Editor of the *British Medical Journal*.

In proposing the health of the Association Professor Robert Platt remarked that, although specialization was inevitable in these days, it should not interfere with the doctor–patient relationship, which belonged to the art of medicine. With so much of science available there was no need for pretence, and doctors could now be frank about what they did and did not know. In his reply the President, Dr. P. Bauwens, was able to report a successful year in the affairs of the Association. The health of the guests was proposed by the Vice-President, Dr. Hugh Burt, and Dr. Ian Grant responded.

At the conclusion of the dinner Professor Platt opened the Library and Censors' Room of the College, so that, as he so disarmingly put it, some could now admire in comfort the magnificent rooms through which they previously had passed in perhaps a less leisurely manner. This privilege was greatly enjoyed by the members and their guests.

D. R. L. Newton

ANNUAL GENERAL MEETING

The Fifteenth Annual General Meeting of the Association was held at the Royal Free Hospital, London, on April 26, 1958, at 10.30 a.m. The President, Dr. P. Bauwens, was in the chair, and approximately 90 members were present.

The minutes of the 1957 Meeting, the report of the Council for 1957–8, and the report of the Honorary Treasurer, together with the balance-sheet to December 31, 1957, were all adopted without dissent. It was agreed that future reports of the Council should contain fuller information concerning the activities and decisions of that body.

Officers.—The following officers were elected for 1958–9:

Vice-President: Dr. H. A. Burt
Honorary Treasurer: Dr. Donald Wilson
Honorary Secretary: Dr. A. T. Richardson
Honorary Editor: Dr. A. C. Boyle

As the result of a ballot the following were elected to fill vacancies on the Council:

Dr. F. S. Cooksey
Dr. D. R. L. Newton
Dr. G. O. Storey

Constitution.—The draft of the new constitution was discussed and approved without amendment, and it was agreed that it should take effect from the conclusion of this meeting.

Shortage of Physiotherapists.—Many members expressed concern at the shortage of physiotherapists. Dr. Richardson stated that this was under active consideration by the Ministry of Health, and the meeting agreed that the matter should be considered by the Council.

The President thanked Miss M. Morris for her invaluable secretarial assistance in organizing the affairs of the Association during the past year.

D. R. L. NEWTON

THE NORTH AND MIDLANDS PHYSICAL MEDICINE CLUB

THE fourth meeting of the North and Midlands Physical Medicine Club was held at Droitwich on Saturday, March 8, 1958, when papers were read by Dr. C. F. Hawkins and Dr. H. T. Fawns. Cases were shown by Dr. W. R. N. Friel and Mr. J. A. James. Miss E. Bolton and her physiotherapy team gave demonstrations in the pool and in the gymnasium.

In spite of the appalling weather conditions twenty-three members and guests attended dinner in the evening. It is hoped to hold the next meeting at Durham in the spring of 1959.

REVIEWS OF BOOKS

CORTISONE THERAPY: MAINLY APPLIED TO THE RHEUMATIC DISEASES. By J. H. Glyn. *Pp. 162. 21s. London: Heinemann. 1957.*

This book, which is "designed principally to help the general practitioner", will be of interest and value to all who use, or intend to use, steroid therapy. It contains excellent chapters on the pharmacological properties and side-effects of the different steroids and on the intra-articular use of hydrocortisone. As Lord Cohen points out in the introduction, not all will approve unreservedly of all the opinions expressed by the author. This particularly applies to the chapter on the practical problems of cortisone therapy, and also to the "General Discussion" with which the book ends. It would perhaps have been wiser if he had confined his thesis entirely to the rheumatic and pararheumatic group of diseases, and had omitted the twelve pages on the use of steroids in other conditions, on which he cannot speak with such authority, and which, if mentioned at all, should have been discussed more fully.

In the appendix there is an extensive bibliography, though there are no references to some of the papers quoted in the text. The appendix also includes a detailed and useful description by Dr. J. G. Bearn of the techniques of intra-articular injection.

HUGH BURT

GOUT. By John H. Talbott. *Second edition. Pp. 205. $6.75. New York and London: Grune and Stratton. 1957.*

This monograph has been completely rewritten, the previous edition, published in 1943, now being out of print.

The author begins with a short account of the history of gout and then discusses the heredity, which is still undetermined. An incidence of over 5% of all cases of arthritis in a special clinic is given, which would appear to be rather higher than that usually found in corresponding clinics in this country. It is stressed that gout can occur in children, and that the earlier the age of onset the more severe the disease is likely to be.

A clear account is given of the aetiology, of purine metabolism, and of the renal exchange of urates. This is followed by a discussion of the pharmacology of the various drugs used in treatment. It is considered that serum uric acid levels of over 6 mg. per 100 ml. in the male or over 5·5 mg. per 100 ml. in the female are essential for a diagnosis of gout, and that this level is maintained irrespective of the stage of the disease. The force of this statement is somewhat diminished when, to illustrate the lower range in the female, a case is quoted with values between 4·5 and 6·0 mg. per 100 ml.

The discussion of the clinical features is excellent, though it is a pity that the illustration of an acute attack in a big toe is so poor when the other illustrations are so good. The only criticism that can be levelled is that the question of hypertension in gout is obscure. On page 113 it is stated that hypertension is probably an associated condition and not a complication, and on page 125 that a "significant percentage" of cases have hypertension.

Treatment is discussed under separate headings for the acute attack and for the intercritical period, and shows a balanced, cautious approach. It is recommended that colchicine should be given for the acute attack in such doses as to cause gastro-

intestinal disturbance. Phenylbutazone is recommended only for acute attacks during initial suppressive therapy with modified colchicine, for the attack inadequately treated with colchicine, or for residual symptoms after the acute attack in preference to a second course of colchicine. It is not recommended for the intercritical period because of its toxic effects. In the intercritical period colchicine is given to every patient with one attack a year, and it is stressed that full doses are still effective for the acute attack should it develop. Hyperuricaemia as such is not treated. Benemid is not used alone, the author stating that he has no experience of this form of treatment—"nor does he propose to obtain this". The reason is obscure, as many physicians in this country have found it effective, at any rate in milder cases. Large tophi are removed surgically to avoid excessive urate load on the kidneys, and the importance of an adequate fluid intake to avoid precipitation is stressed. High purine foods are avoided, but proteins allowed in normal quantities.

This monograph gives a clear, well-written, and balanced account of all aspects of gout and can be cordially recommended to both general physicians and rheumatologists. The treatment given by the author is outlined clearly, but will not meet universal acceptance in this country.

D. C. BEATTY

THE RESPIRATORY MUSCLES AND THE MECHANICS OF BREATHING. By E. J. Moran Campbell, M.D., M.R.C.P. *Pp. 131. 20s. London: Lloyd-Luke (Medical Books) Ltd. 1957.*

In this monograph the author presents an account of the function of the respiratory muscles and the mechanics of breathing, based on his own considerable experimental research and an extensive review of the literature. At the beginning of each chapter is a brief anatomical description of the muscles to be discussed, followed by an account of their mechanical actions. A detailed account of the technical aspects of electromyography as applied to the examination of the muscles of breathing is given in the appendix.

The author aims to make the work intelligible to the non-specialist reader, although those not well acquainted with respiratory physiology may find some of it perplexing. An extensive bibliography has been included, drawing together work published in many different branches of medical literature. Respiratory physiologists and research workers in this field will find this a stimulating and useful work.

JOYCE P. LOMAS